Fibromyalgia is a neuropathic pain syndrome.

Manuel Martinez-Lavin

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*The Journal of Rheumatology* is a monthly international serial edited by Earl D. Silverman featuring research articles on clinical subjects from scientists working in rheumatology and related fields.
Septic Abscess in a Child with Juvenile Idiopathic Arthritis Receiving Anti-Tumor Necrosis Factor-α Therapy

To the Editor:

We read with interest the case report by Kaur and colleagues1 of septic arthritis and abscess formation caused by Actinobacillus ureae in a patient with rheumatoid arthritis receiving etanercept and methotrexate (MTX). There are many reports of infection associated with tumor necrosis factor-α (TNF-α) blocking agents, including those of the superficial skin2 and abscess3-6. There have been post-market reports of etanercept-associated abscesses in the pediatric population with chronic arthritis7. However, none of these cases have been documented in the literature. This includes not even a single case reported in the open-label extension study8 of etanercept for treatment of juvenile idiopathic arthritis (JIA), now through 4 years of followup9. Recently, we cared for a child who developed a deep abscess while on treatment with etanercept and MTX.

An 11-year-old African-American girl was diagnosed with polyarticular JIA 4 months prior to presentation to the emergency department with a fever to 102°F and an 8 x 10 cm area of erythema and tenderness over the left flank. She reported an insect bite to the area occurring the day before. Her current therapy consisted of etanercept 25 mg subcutaneously twice a week (started 2 months before), MTX 25 mg subcutaneously weekly (started 4 months before), prednisone 10 mg orally daily, and naproxen 500 mg orally twice a day. In the emergency department she was given intravenous ceftriaxone, but within several hours of admission the area of tenderness and erythema spread rapidly. A magnetic resonance scan showed heterogeneous abnormal low signal in the subcutaneous flap in T1-weighted images and heterogeneous high signal in T2 (Figure 1). This is consistent with necrotizing fasciitis, and extended from the inferior liver to the iliac crest, roughly 16 cm in total length. She was taken to surgery, where the area was dissected down to the fascia. A localized area of pus in the soft tissue was debried and a penrose drain was placed. A biopsy of the fascia showed evidence of inflammation but not necrosis. She remained hospitalized over the next 5 days with continued serosanguinous drainage from the wound, undergoing antibiotic therapy with vancomycin and clindamycin.

The abscess culture grew methicillin-resistant Staphylococcus aureus sensitive to clindamycin, vancomycin, and trimethoprim-sulfamethoxazole. She was discharged on a 14-day course of trimethoprim-sulfamethoxazole. Etanercept and MTX were stopped during the phase of active infection. After antibiotic therapy was completed, MTX was restarted and etanercept was changed to adalimumab, 40 mg subcutaneously every 2 weeks.

Etanercept appears to be an effective therapy for polyarticular JIA unresponsive to more traditional therapy of intraarticular corticosteroid injections and MTX8,10. However, as noted by Kaur and colleagues, 21% of adverse events attributable to etanercept are infection-related, and physicians must be vigilant in screening for early evidence of infection1. Additionally, Song, et al reported that infliximab, a TNF-α inhibitor not currently approved for treatment of pediatric arthritis, delayed the onset and reduced the incidence and severity of abscess formation induced with S. aureus in a primate model11. Conversely, in the same study, etanercept caused a modest increase in the incidence and severity of abscess formation11. Thus, not all TNF-α inhibitors may share the same risks for infections [e.g., monoclonal antibodies (infliximab and adalimumab) versus soluble TNF-receptor fusion proteins (etanercept)]. Our case of a deep tissue abscess occurring during TNF-α inhibition highlights the importance of close attention to the safety profiles of biologic agents used to treat JIA.

REFERENCES


To the Editor:

Drs. Fitch and Cron describe a case of abscess formation in a child with juvenile idiopathic arthritis who was being treated with prednisone, methotrexate (MTX), and etanercept. Our patient had rheumatoid arthritis with overlap features and developed septic arthritis and abscess formation with *Actinobacillus ureae* while on treatment with MTX and etanercept. We agree with their conclusion to pay close attention to safety profiles of all biologic agents. However, Fitch and Cron raise the possibility that all TNF-α inhibitors do not share the same risks for infections, based on a study of a primate model by Song, *et al*.1 The study showed that administration of anti-TNF-α monoclonal antibody to *S. aureus*-challenged monkeys results in both a delay in the onset and a reduction in the incidence and severity of abscess formation, compared with saline administration1. They also administered etanercept using the same regimen, which resulted in a modest increase in the incidence of abscess formation and no improvement in the severity of abscess formation, compared with saline administration1.

It is hard to predict how these findings will translate to clinical significance in humans, since abscess formation has been reported in patients being treated with all TNF-α inhibitors2-5. Septic arthritis6-7 and osteomyelitis8 have been reported in patients treated with infliximab. Therefore physicians should be vigilant to discern the earliest signs of infections in all patients treated with any of the biologic agents.

PRIMAL P. KAUR, MD, Assistant Professor of Medicine, Division of Rheumatology, Temple University School of Medicine, 3401 N. Broad Street, Philadelphia, Pennsylvania 19140, USA.

E-mail: primal.kaur@tuhs.temple.edu; CHRIS T. DERK, MD, Assistant Professor of Medicine, Division of Rheumatology, Thomas Jefferson University, Philadelphia, PA; MELANIE CHATTERJI, MD, The Arthritis Group, Philadelphia, PA; RAPHAEL J. DEHORATIUS, MD, Professor of Medicine, Division of Rheumatology, Thomas Jefferson University, Philadelphia, PA.

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Hypothyroidism Contributes to Increased Triglyceride Levels Among Patients with Systemic Sclerosis

To the Editor:

We read with interest the article by Kodera and colleagues1 on anti-lipoprotein lipase antibody-mediated lipid metabolism abnormalities in patients with systemic sclerosis (SSc). The report provides new insight into the pathogenesis of dyslipidemia in SSc and probably in other connective tissue disorders. The findings are of special importance in light of the increased prevalence of macrovascular disease observed in this group of patients2. However, according to the results obtained in the study, only one-third of patients with increased triglyceride levels had anti-lipoprotein lipase antibody, which suggests the involvement of other factors. A lipid profile is the result of complex metabolic processes involving dietary habits, environmental influences, race, and endocrine system function. Among the latter, thyroid function is of great interest, with its influence on lipoprotein metabolism.

We describe our experience in lipid metabolism among patients with diffuse SSc. In a cohort of 49 patients with diffuse SSc we observed clinical and laboratory features of hypothyroidism in 13 patients (27%) and increased triglyceride levels in more than 55%. Unexpectedly, no differences were observed between hypothyroid and euthyroid SSc patients with regard to their total cholesterol or HDL and LDL-cholesterol levels. Moreover, the patients with hypothyroidism had statistically significantly higher levels of triglycerides than euthyroid patients with SSc3.

Hypothyroidism is a common finding in patients with SSc. It is estimated that about half of SSc patients experience overt or latent hypothyroidism4. In contrast to previous reports3,5, patients with SSc showed marked lipid metabolism abnormalities, which may predispose them to early development of macrovascular disease and serious cardiovascular complications. SSc has a very strong influence on triglyceride metabolism in all subgroups of patients, since all SSc patients had higher triglyceride levels. Thus, hypothyroidism may partially explain the elevation of triglyceride levels in the patients with SSc. Kodera, et al have provided more information on lipid metabolism in SSc, supporting the effect of autoimmune mediation upon lipoprotein-lipase, a key enzyme in lipid metabolism. Consistent with this finding, hypothyroidism may be responsible for triglyceride elevation in patients who had no anti-lipoprotein lipase autoantibodies. On the other hand, immune-mediated lipoprotein lipase dysfunction could be responsible for increased triglyceride levels in euthyroid patients with SSc. The findings of these 2 studies might offer a way to verify the hypothesis on overlapping anti-lipoprotein lipase antibody and hypothyroidism, and the influence of both conditions on lipid metabolism in patients with SSc.

PRZEMYSŁAW J. KOTYŁA, MD, PhD, Lecturer; MACIEJ LEWICKI, Assistant; EUGENE J. KUCHARZ, MD, PhD, FACP; Professor of Medicine, Chairman, Department of Internal Medicine and Rheumatology, Medical University of Silesia, Ziolowa 45/47, 40-635 Katowice, Poland. E-mail: pkotyla@slam.katowice.pl

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Drs. Kodera and Sato reply

To the Editor:

We are interested in the letter by Dr. Kotyla and colleagues. As described in the discussion section of our article1, we speculate that there are multiple factors that cause dyslipidemia other than anti-lipoprotein lipase antibodies in patients with systemic sclerosis (SSc). It is possible that hypothyroidism may contribute to the elevated serum levels of triglycerides, as noted by Kotyla, et al.

We have seen SSc patients with anti-microsome/thyroglobulin antibodies and/or hypothyroidism. However, our clinical database lacked precise information on this. Nonetheless, a previous study showed that serum anti-thyroid antibodies (antithyroglobulin and/or antimicrosomal antibodies) were detected in 18% and indicated an increased frequency of (sometimes previously unsuspected) clinical and subclinical hypothyroidism in patients with SSc2.

In cases of hypothyroidism, many patients with dyslipidemia have elevations of both serum total cholesterol levels and triglyceride levels3. As shown by Figure 2 in our article4, we found some patients who had both hypercholesterolemia and hypertriglyceridemia. Thus, these patients may suffer from overt or latent hypothyroidism.

MASANARI KODERA, MD, Department of Dermatology, Kanazawa University Graduate School of Medical Science, Kanazawa; SHINICHI SATO, MD, PhD, Department of Dermatology, Nagasaki University Graduate School of Biomedical Sciences, 1-7-1 Sakamoto, Nagasaki 852-8501, Japan. E-mail: s-sato@net.nagasaki-u.ac.jp

Corrections


Tannenbaum H, Bombardier C, Davis P, Russell AS, for the Third Canadian Consensus Conference Group. An evidence-based approach to prescribing nonsteroidal anti-inflammatory drugs. Third Canadian Consensus Conference. J Rheumatol 2006;33:140-57. Please note a slide rule for calculating creatinine clearance (as shown) may be obtained upon request from: creatinineclear@aol.com, not creatinineclear@aol.com, as stated on page 145, column 2, line 5. We regret the error.
Figure 1. An easy-to-use device for calculating creatinine clearance. Available upon request from: creatinineclear@aol.com